AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 99:455–472 (1996)

Anemia and the Transition of Nomadic Hunter-Gatherers to a Sedentary Life-Style: Follow-Up Study of a Kalahari Community

SUSAN KENT AND DAVID DUNN
Anthropology Program, Old Dominion University, Norfolk, Virginia 23519 (S.K.); School of Pathology, South African Institute for Medical Research, University of Witwatersrand, Johannesburg 2000, South Africa

KEY WORDS ESR, Anemia, Chronic disease, Serum ferritin, Iron, Diet, Demography

ABSTRACT Iron profiles of communities of hunter-gatherers and former hunter-gatherers conducted between 1969 and 1987 at Dobe in the Kalahari Desert of Botswana exhibited pronounced differences during periods of rapid culture change. The loss of good health and particularly the increase in anemia through time was attributed to notable changes in diet, although changes in mobility patterns were considered a secondary cause. In 1988 and 1989, studies were conducted at Kutse, also in the Kalahari Desert of Botswana, to ascertain the frequency of anemia at a recently sedentary community in which residents still relied primarily on wild animals for meat. Although not identical, the hematological presentation in 1989 was similar to that in 1988. The studies together suggest that our findings characterize the pattern of health and disease at Kutse, which is unrelated to any specific year or to diet. Additional measures of disease, specifically ESR (erythrocyte sedimentation rate) and oral temperatures, support an interpretation of anemia of chronic disease as the cause of hypoferremia at Kutse. Morbidity is high, in spite of adequate diets, because the residents are transitional from a nomadic to a sedentary lifestyle and from a relatively dispersed to an aggregated settlement pattern. These changes have introduced new health problems.

Over the past decade there has been much disagreement concerning the prevalence and cause of anemia in recently sedentary Basarwa (Bushman, San) communities. Studies conducted between 1981 and 1988 at various sedentary Kalahari communities indicate a high level of anemia, but researchers disagree whether the anemia is dietary-induced iron deficiency or the anemia of chronic disease/infection (Fernandes-Costa et al., 1984; Kent and Lee, 1992) For example, two separate research groups (Hansen et al., 1993, and Kent and Lee, 1992) examining the same data collected in 1987 reached opposite conclusions on even the presence of anemia, much less on its cause. The following research was conducted to determine the etiology of high levels of anemia found in 1988 in a recently sedentary Kalahari community.

ETIOLOGY OF ACQUIRED ANEMIA: DIET OR CHRONIC DISEASE?

As is well known in medicine, the etiology of acquired anemia is difficult, if not impossible, to determine in the absence of serum ferritin values or bone marrow aspirations (e.g., Mohler, 1992). The reason for this difficulty is that both iron deficiency anemia and

Received February 23, 1990; accepted September 25, 1995.

Address reprint requests to Dr. S. Kent, Anthropology Program, Old Dominion University, Norfolk, VA 23529.

David Dunn is presently at 19 Conifer Place, Beechboro, 6063 Perth, Western Australia.

TABLE 1. Comparison of anemia of dietary iron deficiency and anemia of chronic disease

Anemia	Hemoglobin	Transferrin saturation	Serum iron	ESR	Serum ferritin
Iron deficiency	Decreased	Decreased	Decreased	Normal	Decreased
Chronic disease	Decreased	Decreased	Decreased	Increased	Normal-raised

Modified from Kent (1992).

the anemia of chronic disease/infections are characterized by hypoferremia as indicated by subnormal levels of hemoglobin, hematocrit, serum iron, and/or transferrin saturation (see Table 1). Consequently, serum ferritin, an indirect measure of iron stores, and bone marrow examination of iron stores are the only conclusive means for distinguishing between the two anemias (for reasons why and their evolutionary importance, see Kent et al., 1994). Subnormal serum ferritin values (below 12–15 μg/L) indicate subnormal iron stores while the reverse, normal to evaluated levels, indicate normal to elevated iron stores in the marrow (Burns et al., 1990). In fact, the definition of the anemia of chronic disease is the presence of hypoferremia (low iron levels as indicated by below normal values in one or more of the iron indices in Table 1) in combination with normal to elevated serum ferritin levels.

HISTORY OF JU/'HOANSI BASARWA MEDICAL RESEARCH

In 1969, Ju/'hoansi (referred to as !Kung in the past; Biesele, 1993; Lee, 1993) Basarwa were still nomadic hunter-gatherers who subsisted primarily on a diet of wild plants and animals (Kent and Lee, 1992). A hematological study (Metz et al., 1971) of Ju/'hoansi at Dobe, located in the northwestern part of the Kalahari Desert of Botswana (Fig. 1), revealed a health status described as very good. The presence of anemia was remarkably low for all subgroups of the population. The absence of anemia in pregnant women and among the few individuals afflicted with hookworm and other parasites (e.g., malaria) was attributed to the group's nomadic mobility pattern, hunting-gathering lifestyle, and the use of cast iron pots for cooking, which provided large amounts of exogenous iron (Metz et al., 1971). During the ensuing years, however, the Dobe Basarwa became sedentary, aggregated, and dependent on wage work, livestock (mostly goats and sheep with a few head of cattle), farming, and government assistance programs (Draper and Kranichfeld, 1990). As a consequence of these and other changes at Dobe, their diet differed substantially from the traditional one. By 1987, the Ju/'hoansi diet still included meat, although in contrast to the 1960s, half came from domesticated as opposed to wild animals (Kent and Lee, 1992). Domesticated plants and government-provided flour replaced most of the wild plants traditionally consumed.

Chum!kwe, a sedentary Namibian community composed of displaced Nyae Nyae and other Ju/'hoansi, was studied in 1981 (Fernandes-Costa et al., 1984). Hematological studies revealed the presence of both dietary deficiency and poor health as a result of the residents' abandonment of traditional subsistence resources, reliance on alcohol, and general disruption of traditional life. Approximately 47% of the Chum!kwe children had subnormal hemoglobin levels and 33% had subnormal serum ferritin levels (Fernandes-Costa et al., 1984). Parasites, which compete with the host for iron and can cause gastrointestinal bleeding, were detected in 23% of the fecal samples obtained. The most common parasites identified were hookworm (*Necator americanus*) and giardia (*Giardia lambia;* Fernandes-Costa et al., 1984). Although the ages of those infected by parasites were not noted, children are often more susceptible to such infections than are adults due to their behavior. Fewer adults had subnormal Hb levels than did children—below normal values were present in 10% of the males and 12% of the females (Fernandes-Costa et al., 1984). Significantly, no adult males and only 6% of adult females had subnormal serum ferritin levels, indicating the absence of iron deficiency anemia in most cases. Despite these findings, the authors of the study implicated iron deficiency as the

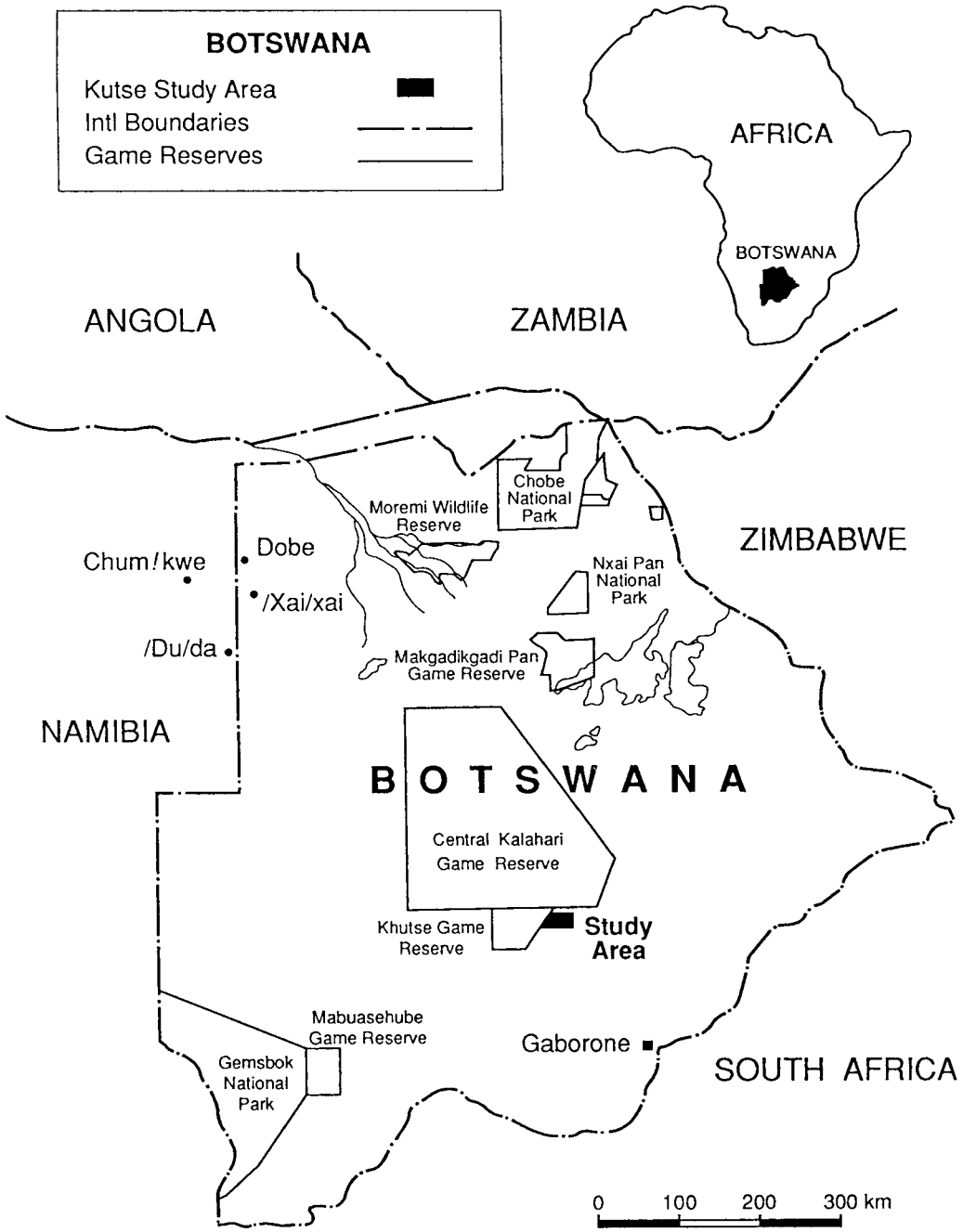

Fig. 1. Location of the Kutse community.

major cause of anemia for all groups (Fernandes-Costa et al., 1984). In contrast, other researchers suggested that the presence of anemia resulted primarily from chronic disease/infection for the adults, and from possibly parasites, chronic disease, and, in some cases, dietary iron deficiency for the children (Kent and Lee, 1992).

In 1988, we conducted a hematological study at the recently sedentary Kalahari community of Kutse to determine the effect of diet and demography on anemia (Fig. 1). Because of historical circumstances, Kutse residents remain primarily hunters-gatherers, although not exclusively so (Kent, 1996). Unlike the 1987 Dobe and 1981 Chum!kwe studies where diet had changed dramatically, the Kutse consumption of meat was more or less similar to the diet of the 1969 Dobe study volunteers. The Kutse studies allow us to examine the effect of sedentism and aggregation by keeping diet relatively constant between the Dobe 1969 and the Kutse 1988 and 1989 studies, while varying mobility.

1988 AND 1989 STUDIES: BACKGROUND AND METHODS

The Kutse population fluctuates depending on the day, season, and year. Not including visitors, around 128 to 153 persons occupy Kutse at any one time. The number of inhabited camps is as variable, depending on the time of the census but range from 20 in 1987 to 23 in 1988 to 24 in 1989. As with the other demographic figures, the precise number of individuals occupying a single camp is difficult to specify and often in flux, particularly because of the common practice of visiting within the Kutse community and the presence of non-resident visitors. Generally camps are inhabited by somewhere between five and 20 people. Frequent non-resident visitors, who may stay from 1 day to 3 months or more at a single camp, increase this number by anywhere from one to 10 persons.

The 1988 hematological study indicated that there was no significant difference in the hematology, morbidity, or diet between the Basarwa (Bushmen; San) and the Bakgalagadi (Bantu-speakers) who both occupy Kutse. Therefore, both ethnic groups are viewed here as a single study populations. As a consequence of adding both groups together, the numbers presented in Kent and Dunn (1993) differ slightly from those reported here. The 1989 study added two measures of chronic disease/infection to the battery of tests: erythrocyte sedimentation rate (ESR) and oral temperature as measured with a digital thermometer. These tests were in addition to the complete blood count, serum ferritin, chemistry panel, and other hematological indices investigated (Tables 2 and 3). In order to maintain consistency, both the 1988 and 1989 hematological studies were conducted during the first week in August.

Detailed dietary information was collected through observation and formal interviews from May to August in both 1988 and 1989. The senior author lived at three different camps during 1988 and 1989. Each camp was composed of one to two households of nuclear and extended families. All food consumed by inhabitants over a 24-hour period was recorded. Who ate what, the person's age and relationship to the head of the household, what others were doing at the time, and how the food was obtained and processed were recorded for each consumption event. Questions concerning food consumption while the senior author was away from camp spot checking diets elsewhere in the community, conducting interviews, or otherwise collecting data were asked upon return to the focus camp. At least 2 days a week were spent continuously at the focus camp (e.g., weekly continuous 48-hour or longer period was observed). The Appendix lists the days meat was consumed at the different camps in which the senior author lived during the dry season of 1989 just before the health/hematological studies were conducted.

Permission to perform the 1988 and 1989 hematological studies at Kutse was granted by the Office of the President, Gaborone, Botswana, May 18, 1988. Each volunteer gave his or her permission for the study prior to being examined and having blood drawn. In the case of children, permission was obtained from their parents and/or from the older children themselves. Infants and toddlers did not have blood drawn because it was felt that they were too young to under-

TABLE 2. Kutse hematology dry seasons 1988 and 1989

	Hematocrit[1] N (%)	ESR[2] N (%)	Serum ferritin[3] N (%)	Serum protein[4] N (%)	Serum folates[5] N (%)	B_{12}[6] N (%)
1989 Males						
Normal	19 (86.4%)	11 (50.0%)	14 (63.6%)	19 (86.4%)	11 (50.0%)	16 (72.7%)
Below normal	2 (9.1%)	— —	— —	— —	— —	— —
Above normal	1 (4.5%)	11 (50.0%)	8 (36.4%)	3 (13.6%)	11 (50.0%)	6 (27.3%)
1989 Females						
Normal	30 (96.8%)	18 (58.1%)	24 (80.0%)	23 (76.7%)	13 (41.9%)	21 (67.7%)
Below normal	1 (3.2%)	— —	— —	— —	— —	— —
Above normal	— —	13 (41.9%)	6 (20.0%)	7 (23.3%)	18 (58.1%)	10 (32.3%)
1989 Children						
Normal	28 (82.3%)	11 (32.4%)	28 (82.4%)	28 (90.3%)	17 (50.0%)	15 (44.1%)
Below normal	6 (17.7%)	— —	— —	— —	— —	— —
Above normal	— —	23 (67.6%)	6 (17.6%)	3 (9.7%)	17 (50.0%)	19 (55.9%)
1988 Males						
Normal	22 (75.9%)		15 (50.0%)	12 (70.6%)	16 (66.7%)	23 (95.8%)
Below normal	7 (24.1%)		— —	— —	1 (4.2%)	— —
Above normal	— —		15 (50.0%)	5 (29.4%)	7 (29.2%)	1 (4.2%)
1988 Females						
Normal	36 (83.7%)		37 (86.0%)	32 (91.4%)	16 (43.2%)	33 (89.2%)
Below normal	6 (14.0%)		— —	— —	— —	— —
Above normal	1 (2.3%)		6 (14.0%)	3 (8.6%)	21 (56.8%)	4 (10.8%)
1988 Children						
Normal	37 (86.1%)		36 (94.7%)	32 (91.4%)	12 (36.4%)	24 (72.7%)
Below normal	5 (11.6%)		— —	— —	— —	— —
Above normal	1 (2.3%)		3 (5.3%)	3 (8.6%)	21 (63.6%)	9 (27.3%)

[1] Normal ranges: males 41–54%; females 37–49%; males aged 12–15 years 37–49%; females aged 12–15 years 36–46%; children aged 7–11 years, 35–45%; children aged <6 years 34–44%.
[2] Normal ranges: males 0–9 mm/hr; females 0–20 mm/hr; children 0–13 mm/hr.
[3] Normal ranges: adults 15–240 ng/ml; children 15–140 ng/ml.
[4] Normal ranges: adults 60–80; children 60–80.
[5] Normal ranges: adults 3–14 ng/ml; children 3–14 ng/ml.
[6] Normal ranges: adults 200–1,100 pg/ml; children 200–1,100 pg/ml.
Numbers differ slightly between the 1988 figures reported here and those reported in Kent and Dunn (1993) because both ethnic groups the basarwa and the Bakgalagadi have been combined in this table for the 1988 and 1989 data. The same normal ranges are used in both articles.

stand, coupled with our belief that it may not be ethical to subject them to a blood test in a research setting. Children's ages were established on the basis of standard physiological developmental sequences (methods used were similar to those employed among the Ju/'hoansi by demographers in the 1960s; e.g., Howell, 1979). Adults' ages were estimated according to their approximate ages during known events (e.g., their age at someone's birth), their appearance, and the number and ages of their children. Adults were grouped by age into 10- to 20-year categories. A t-test showed no significant difference in the ages of either men or children between the 1988 and 1989 studies. The women tested in 1989 were significantly younger than those in 1988, but both means were well within the age of menstruating women; therefore, neither study included a larger proportion of menopausal women which could alter values and make means difficult to compare between years (mean age for females in 1988 was 39.9 and for 1989 was 33.6 years).

Brief physical examinations were conducted for both years (1988 and 1989) just before blood was drawn. These examinations were based on a standard checklist that included lungs, heart, abdomen, mouth, throat, and skin. Because some residents were foraging away from Kutse or were visiting other villages, not everyone was available for study. During the 1988 study, 127 Kutse residents were examined, representing 98% of the community. In 1989, 108 persons, or 71% of the entire Kutse community, were examined. Individuals who were not examined were absent from the community when the study was conducted. Those examined but did not have blood drawn were very young children. No health differences were detected in 1988 or 1989 between those volunteers who provided blood and those who did not.

The 1988 hematological study has been

TABLE 3. Leukocytes values (WBC and differential) at Kutse during the dry seasons of 1988 and 1989

	WBC[1] N (%)	Monocytes[2] N (%)	Lymphocytes[3] N (%)	Eosinophils[4] N (%)
1989 Males				
Normal	19 (86.4%)	20 (90.9%)	9 (40.9%)	16 (76.2%)
Below normal	1 (4.5%)	— —	6 (27.3%)	— —
Above normal	2 (9.1%)	2 (9.1%)	7 (31.8%)	5 (23.8%)
1989 Females				
Normal	27 (87.1%)	24 (85.8%)	11 (39.3%)	17 (60.7%)
Below normal	2 (6.5%)	— —	— —	— —
Above normal	2 (6.5%)	4 (14.3%)	17 (60.7%)	11 (39.3%)
1989 Children				
Normal	34 (100%)	28 (84.8%)	18 (54.6%)	5 (15.2%)
Below normal	— —	— —	6 (18.0%)	— —
Above normal	— —	5 (15.2%)	9 (27.0%)	28 (84.8%)
1988 Males				
Normal	22 (73.3%)	18 (64.3%)	13 (46.4%)	18 (64.3%)
Below normal	— —	— —	6 (21.4%)	— —
Above normal	8 (26.7%)	10 (35.7%)	9 (32.1%)	10 (35.7%)
1988 Females				
Normal	38 (88.4%)	28 (65.1%)	22 (51.2%)	34 (79.1%)
Below normal	— —	— —	9 (20.9%)	— —
Above normal	5 (11.6%)	15 (34.9%)	12 (27.9%)	9 (20.9%)
1988 Children				
Normal	42 (97.6%)	20 (50.0%)	14 (35.0%)	14 (35.0%)
Below normal	— —	— —	16 (40.0%)	— —
Above normal	1 (2.4%)	20 (50.0%)	10 (25.0%)	26 (65.0%)

[1] Normal ranges: adults 4,000–11,000; children aged 8–14 years 4,500–13,500; children aged 4–7 years 5,000–14,700 children aged <3 years 5,500–15,500.
[2] Normal ranges: adults 0–7; children aged 7–14 years 0–6%; children aged <7 years 0–5%.
[3] Normal ranges: adults 25–36%; children aged 6–14 years 33–50%; children aged <6 years 32–52%.
[4] Normal ranges: adults 0–4%; children 0–3%.
Numbers differ slightly between the 1988 figures reported here and those reported in Kent and Dunn (1993) because both ethnic groups have been combined in this table, as they were for the 1989 data. In addition, the differential white blood cells were presented as absolutes in Kent and Dunn (1993) and here they are percents.

described in detail (Kent and Dunn, 1993). In 1988, the Basarwa study population consisted of 106 individuals—39 adult females, 25 adult males, and 42 children under the age of 15. Not included in these figures, or in the analysis reported in Kent and Dunn (1993), were 10 Bakgalagadi Bantu-speakers. The 1989 hematological study consisted of 87 individuals—31 adult females, 22 adult males, and 34 children under the age of 15 years. By chance, there were fewer people in Kutse during the days of the study in 1989 than were present during the 1988 study; however, no differences could otherwise be discerned between the two study populations. A total of 72 people in the 1989 study (i.e., 83% of the 1989 group) also were volunteers in the 1988 study.

Oral temperatures of volunteers were measured at Kutse on July 26, 1989, between 11:00 A.M. and 5:00 P.M. (temperatures were not systematically measured in 1988). The day was selected randomly 1 week prior to the hematological tests and physical exams. A digital thermometer was used with a protective plastic covering that was replaced after each measurement. Temperatures were taken while people were lounging around campfires (the study was conducted during the cold dry season). Children were requested to sit before temperatures were measured in order to obtain an at-rest measurement. Individuals were requested to abstain from smoking homemade metal or bone pipes before their temperatures were measured.

Residents' perceptions of illness were recorded through observation and formal interviews from May to August in both 1988 and 1989. At Kutse, the appropriate reply when greeted entails a short statement on one's health. Such statements are followed up with questions concerning the individual's health and reasons for it, all of which were recorded (in many cases, complaints were of "body sickness," usually meaning a strained muscle or backache; these were recorded but not classified as illness). In addi-

tion, the senior author was summoned to any camp in which a very ill or injured person was located. Other people in need of first aid, but not seriously incapacitated, would seek out the senior author for treatment. These encounters also were recorded.

Blood was collected from the antecubital vein into vacutainer tubes in 1988 and 1989 by the same laboratory technician. Blood for the full blood count was drawn into sequestrine (EDTA) tubes and stored at 4°C until tested at the South African Institute for Medical Research Laboratory in Johannesburg 3 to 4 days later. Blood was drawn between approximately 8:30–9:00 A.M. and 4:00 P.M. (some tests, such as serum iron, have significant shifts in values due to diurnal cycles).

Hematocrits were measured in the field with a generator-operated centrifuge within 4 to 6 hours after collection during both 1988 and 1989. Clotted blood samples both years were centrifuged in the field. Serum was separated and stored in liquid nitrogen at −170°C until tested 3 to 7 days later in the Johannesburg laboratory. The blood, except for hematocrits and ESR which were measured in the field, was also analyzed at the laboratory.

In both 1988 and 1989, hemoglobin concentrations and red cell counts were measured in a Coulter Model S Senior Counter (Hialeah, FL) using 4C control. Blood smears were prepared at the time of blood collection. Thin films were fixed in methanol in the field; later they were stained and examined in the laboratory for red cell morphology and white cell differential count. Thick films were stained with Giemsa's stain and examined for malaria in the laboratory in 1988 and 1989. Serum ferritin was measured by radioimmunoassay (Amersham, U.K.), serum iron by a colorimetric method (Iron Test Roche, Roche, Burlington, NC) using a Microcentrifugal Analyzer III (Instrumentation Laboratories), and serum transferrin by an immunoturbimetric method (Dako) using the Microcentrifugal Analyzer III. Blood chemistries for both years were measured with a "SMAC" autoanalyzer (Technicon, Tarrytown, NY).

ESR was measured in 1989 using the Wintrobe method (it was not measured in 1988). Blood was collected in EDTA vacutainer tubes and then poured into the Wintrobe tubes. The value of each sedimentation rate was recorded at the meniscus of the red cells after precisely 1 hour. Standards of normalcy established worldwide and used in the literature were employed in this study.

Most multiyear studies employ different laboratories for test analyses, which can cause variability in test results simply due to the use of different procedures or equipment. In order to maintain consistency among the various studies, our 1988 and 1989 Kutse analyses were conducted at the same laboratory, often by the same laboratory technicians and equipment, as were the previous 1969 and 1987 Dobe and the 1981 Chum!kwe studies. The 1969 serum iron measurements had to be converted to the System International since micromoles/liter is currently used. Vitamin B_{12} used different measures in 1969, although the actual values are numerically identical, except for lower normal limits which changed slightly. These were accounted for (see Kent and Lee, 1992, for a complete comparison between the 1969 and 1987 Dobe studies). Moreover, all analyses were conducted under the supervision of Dr. Trefor Jenkins at the Laboratory for Medical Research using the same reference standards. As a consequence, differences between studies are not due to laboratory variations.

SUMMARY OF 1988 RESULTS

Of the 106 Kutse resident volunteers examined during 1988, 50–52%, depending on the subpopulation, had subnormal serum iron values and 35–49% had subnormal transferrin saturation levels. No one, Basarwa or Bakgalagadi, had subnormal serum ferritin levels in 1988. In fact, 92% of the hypoferremic adult Basarwa had serum ferritin levels greater than 50 μg/L, which is a level indicative of the anemia of chronic disease/inflammation. This finding was corroborated by the lack of protein deficiency in any of the blood chemistries. In contrast, serum protein was elevated in 9 to 29% of the groups tested. A relatively large number of adults had elevated white blood cell counts and, of those, 58% were also hypoferremic.

At the same time, 92% of all age, sex, and ethnic categories had normal hemoglobin levels. However, hemoglobin values are not very diagnostic because they may be increased by smoking, a habit in which, with very few exceptions, all Kutse adults and many older children indulge (Centers for Disease Control, 1989).

Elevated vitamin B_{12} levels were found in 14 individuals; no subnormal values were found. Only one individual had a serum folate deficiency. The blood chemistries yielded no evidence of severe nutritional deficiencies. An elevated mean number of absolute eosinophil counts were found in 41 individuals. The high prevalence of eosinophilia is consistent with the possible presence of helminthic or other parasites. Stool samples were not obtained because the subjects were reluctant to provide them and we did not want to pressure them to do something that they felt was offensive. Of those with elevated eosinophil counts, 72% of the adults and 71% of the children were also hypoferremic, although all had normal to elevated serum ferritin levels. Elevated monocyte counts were found in 31 individuals (29 to 40% of those tested); abnormal lymphocyte counts were found in 5 to 21%.

No malaria was identified in any of the blood smears, even though a few individuals in earlier studies among Ju/'hoansi at Dobe were afflicted with malaria. Kutse is located in a much drier part of the Kalahari Desert where malaria has not been reported. We tested for malaria to demonstrate the validity of our assumption of its absence and to replicate as much of the Dobe studies as possible.

1989 RESULTS

Because of the remote location of the study community and its distance to a research laboratory, lysis occurred in some samples during transportation. We do not include any of the potentially contaminated data in the analysis reported here. We instead use the results of measurements performed in the field, such as hematocrits and ESR, as well as those indices not greatly impacted by lysis.

Of the children tested, 18% had subnormal hematocrits in 1989 (Table 2); fewer adults had subnormal hematocrits (9% of the males and 3% of the females; Table 2). None of these individuals had subnormal serum ferritin values, indicating the absence of iron deficiency (or dietary-induced) anemia. Instead, elevated serum ferritin values were found in 18 to 36% of the subgroups (Table 2).

Perhaps of greater importance were the ESR results. ESR is an acute phase reactant that is strongly associated with the presence and severity of a variety of infectious and inflammatory diseases (Del Beccaro et al., 1992; Johansson, 1992). Because ESR is a non-specific indicator of disease, its utility in clinical practice has been questioned; however, its use in determining the presence or absence of chronic disease is quite valuable for reasons discussed below. ESR was elevated in 50% of the 22 men tested, 42% of the 31 women, and 68% of the 34 children (Table 2; Fig. 2).

White blood cell counts (WBC) were either elevated or subnormal (both can indicate infectious disease) in 14% of the men and in 13% of the women, but not in any children. A large proportion of Kutse individuals in 1989 had elevated eosinophils, ranging in frequency from 24 to 85% of the subpopulations. Eosinophilia indicates the presence of parasites, hematopoietic diseases, various infectious diseases, gastroenteritis, neoplasia (cancer), or miscellaneous skin diseases (Wallach, 1983). Lymphocytes also were abnormal in a large proportion of people. Levels of serum protein, an acute phase reactant associated with disease, were elevated in 10 to 23% of those tested.

Most individuals had normal to elevated serum folate concentrations, indicating diets sufficient in folates (Table 2). An increased number of persons had elevated vitamin B_{12} values in 1989. Some had impressively high levels, and the 1989 mean was significantly higher for all subpopulations than the means obtained in 1988 (Table 4). The overall globulin mean was 31.7 g/L (well within the normal range of 20–35 g/L) and the mean globulin/albumin ratio was 0.72 ($n = 85$) (see Nurse and Jenkins, 1977). As was the case with the 1988 study population, no protozoa (e.g., malaria) were identified in any of the blood smears.

Oral temperatures provide another measure of morbidity and the senior author took

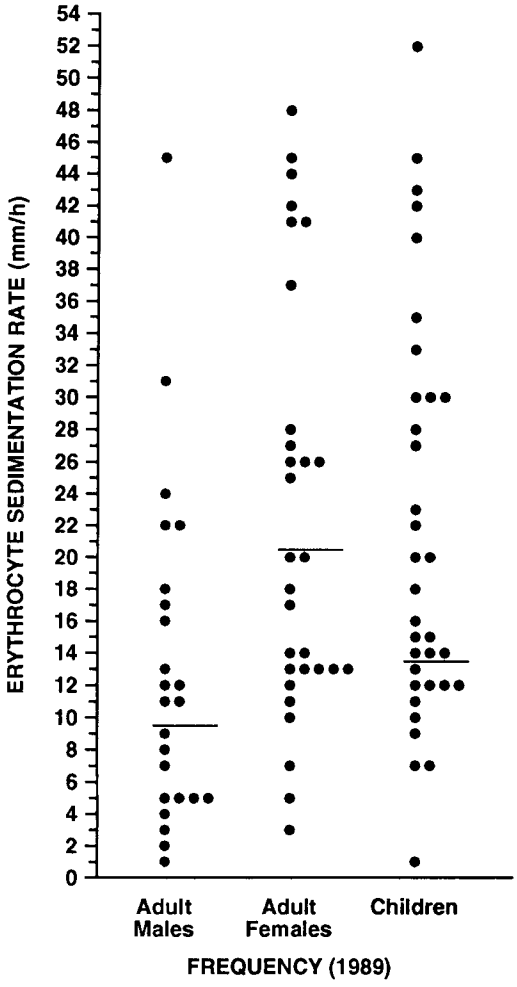

Fig. 2. ESR distribution at Kutse; individuals (dots) distributed above the horizontal line have elevated values.

everyone's temperature in Kutse on a randomly selected day approximately 1 week before the hematological study was conducted. Only those camps occupied on July 26, 1989, are shown on the map in Figure 3. Those investigated consisted of 12 of the 24 camps inhabited during the entire 1989 study season (some people were gone for the day or longer, and their camps are not included in Figure 3). Six camps not examined were temporarily abandoned when the senior author visited them, and she did not have time to return later that day when the occupants had returned to camp. Ten of the 12 camps investigated had at least one individual with a temperature ≥37.2°C (98.9°F). Twelve persons of the 24 tested, or 50%, had a temperature ≥37.2°C. Temperatures ranged from 35.8°C to 38.0°C (96.5°F to 100.3°F). All individuals who said they were not ill did *not* have an elevated temperature, indicating greater validity for interpreting the findings as supportive of the clinical and hematological data. While some people who claimed to be sick also did not have an elevated temperature, their belief of ill health was based on the perception that sore muscles resulting form physical strains constitute "illness." These findings support the clinical and hematological data based on the Western definition of illness.

Clinical observations

As noted in 1988, residents perceived a high morbidity at Kutse. Fever, congestion, coughs, generalized pain, fatigue, diarrhea, and indigestion were the most commonly reported symptoms. Residents perceived a high level of morbidity in the community, but for unknown reasons. Brief physical examinations revealed that 39% of the volunteers had signs of acute or chronic infection, including palpable lymph nodes, fever, and/or congestion. Of those exhibiting clinical symptoms of infection, 63% (26) were children under the age of 15 years. Most common were respiratory infections, favus skin infections (a fungus also known as witkop or *Trichophyton schoenleinii*), tonsillitis, and otitis media. The most frequent infection overall was respiratory in nature, although among the children, moderate to severe favus skin infections were most common. A number of older individuals had chronic obstructive airway disease (probably in part from smoking). Tuberculosis and other specific respiratory diseases could not be identified because of the remote field conditions of the study population and the difficulty of transporting equipment.

Diet

Kutse hunter-gatherers in 1988 and 1989 had a subsistence strategy similar to the healthy 1969 nomadic Dobe foragers while at the same time had a mobility pattern simi-

TABLE 4. Hematology means of 1989 and 1988 Kutse studies

Year/ group	Hematocrit (%)	WBC (cells/mm)	Serum folate (μM)	B_{12} (g/dl)	Serum ferritin (ng/ml)
			Excluding pregnant women		
1989 Kutse					
Males	45.9 (0.7)	7.0[1] (0.5)	16.1 (2.5)	939.5[1] (74.0)	288.37 (50.1)
Females	41.2 (0.5)	7.4[1] (4.2)	20.6 (2.9)	1149.8[1] (135.2)	166.66 (19.2)
Children	38.4 (0.6)	8.7[1] (0.4)	16.9 (1.7)	1183.0[1] (71.6)	98.4[1] (8.5)
1988 Kutse					
Males	44.1 (0.9)	9.7 (0.7)	11.2 (0.7)	737.5 (52.0)	277.27 (39.1)
Females	41.5 (0.6)	13.0[1] (0.1)	17.1 (0.9)	770.88 (49.9)	150.96 (24.9)
Children	38.4 (0.6)	10.0 (0.3)	17.1 (1.1)	889.50 (64.6)	59.2 (6.0)
			Pregnant women only		
1989 Kutse					
N = 1	37.5 (—)	7.9 (—)	6.8 (—)	976.0 (—)	217.0 (—)
1988 Kutse					
N = 7	37.4 (1.9)	8.5 (7.5)	9.3 (1.7)	760.96 (9.3)	98.0 (19.0)

Values are the mean (SE).
[1]A statistically significant difference between 1988 and 1989 exists within the subgroup where $P \leq 0.05$ (see text for precise $P \leq$ values). Geometric mean for serum ferritin values are 1988: males (n = 30) 195.5 ng/ml; non-pregnant females (n = 43) 102.9 ng/ml; pregnant females (n = 7) 76.7 ng/ml; children (n = 38) 51.1 ng/ml; 1989: males (n = 22) 212.5 ng/ml; non-pregnant females (n = 30) 139.6 ng/ml; children (n = 34) 87.7 ng/ml. Also note that values differ slightly between that presented here and that presented in Kent and Dunn (1993) because here all ethnic groups are combined and in the previous study they were analyzed separately.

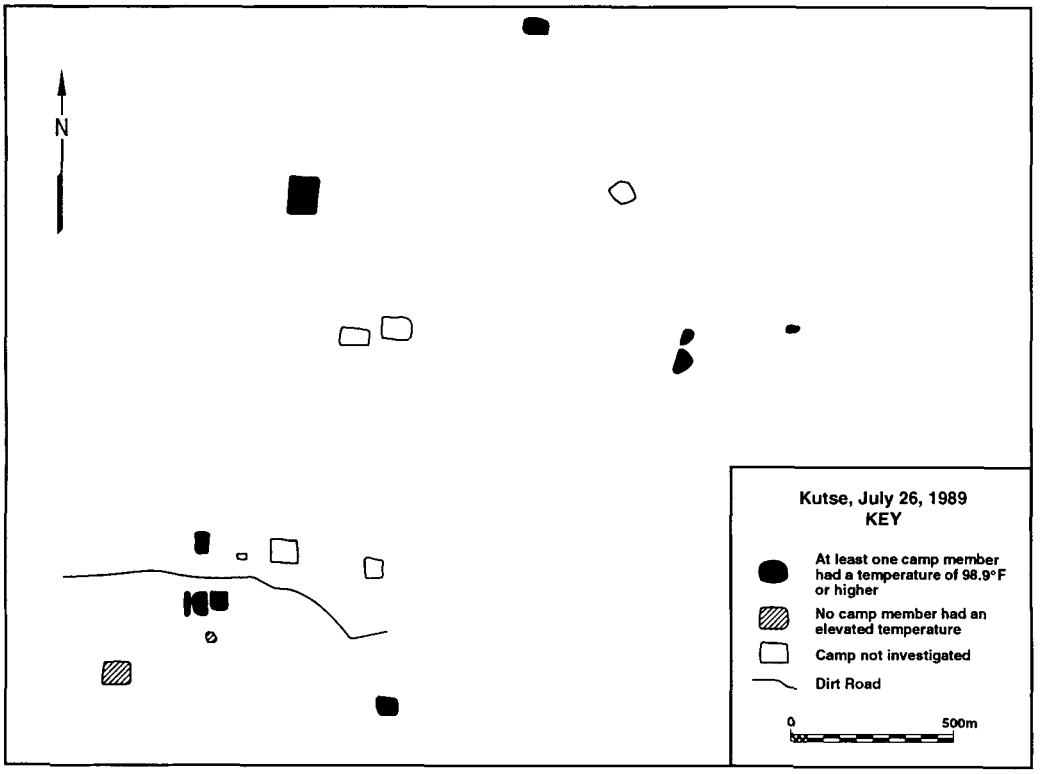

Fig. 3. Map of camps at Kutse during the recording of temperatures on July 26, 1989. Those camps not examined were temporarily abandoned when the senior author visited them.

lar to the 1987 sedentary Dobe community. The majority of families in 1989 consumed meat at least two to three or more times a week (Appendix). With the exception of 2 of 59 days of continuous observations, all meat consumed was from wild animals (on the two exceptions, goat meat obtained from other camps was eaten; Appendix). Wild game included scrub hare (*Lepus saxatilis*), wild cat (*Felis lybica*), bat-eared fox (*Octocyon megalotis*), steenbok (*Raphicerus campestris*), duiker (*Sylvicapra grimmia*), and gemsbok (*Oryx gazella*). Relief food distributed by the government in both 1988 and 1989 consisted primarily of maize flour (mealie meal), beans, and a soy flour mixture. These foods were supplemented with a variety of wild plants, such as melons, tubers, and berries. Wild plants are available throughout the year, although the species and densities change seasonally. Traditionally, as well as today at Kutse, wild plants are consumed during all seasons (see Silberbauer, 1981, for a discussion of wild plants and seasonality in the Central Kalahari).

Goat milk was a very minor component of some families' diets. Water during both years was obtained from the same borehole (well) located within a 45-minute to an hour's walk from camp, depending on the location of the camp, although traditionally, all liquids at this time of the year were obtained from wild melons and wild animals (e.g., blood and stomach contents; Silberbauer, 1981). Among the Ju/'hoansi in 1969 and 1987, in contrast, water at this time of the year was available at the Dobe and other pans.

1988–1989 STUDY COMPARISONS

Some test result variations between the years 1988 and 1989 are to be expected, simply due to the natural fluctuation of morbidity on any given day in any given month of the dry season. Approximately the same percentages of women and children had abnormal WBC levels in 1988 as in 1989 (Table 3), but the same was not the case for men. About 13% more adult males had abnormal WBC counts in 1988 than in 1989 (Table 3). It must be noted, however, that WBC counts are not reliable predictors of disease. In a study designed to investigate the clinical value of ESR and WBC (among other indices), researchers found that ESR was elevated initially in 92% of children with confirmed acute hematogenous osteomyelitis but: "The WBC count was a poor indicator of acute hematogenous osteomyelitis, since only 35% of the children had leukocytosis (WBCs $> 12 \times 10^9$/L) at the time of admission" (Unkila-Kallio et al., 1994:59).

While more individuals had WBC values within normal range in 1989 than in 1988, a much higher percentage of individuals in all groups had elevated eosinophils (from 24 to 85% per subpopulation in 1989, compared to 21 to 65% of the subpopulations during 1988). As noted earlier, eosinophilia is associated with a variety of parasitic, bacterial, viral, and neoplastic diseases. During both years, lymphocytes were commonly either above or below normal ranges (in 1988 only 35 to 51% of the subpopulations had lymphocytes within normal range; in 1989, only 39 to 55% did). Causes of abnormal lymphocytes include a wide range of infectious viral and bacterial diseases (Fishback 1992).

Mean hematocrit levels for the two years were compared. A t-test revealed no statistically significant difference between the studies for men ($P = 0.1429$), women ($P = 0.8627$), or children ($P = 0.6459$). We believe that the similarity in hematocrit means between 1988 and 1989 shows that residents were similar in their health status. There was no significant difference between 1988 and 1989 serum ferritin levels for men ($P = 0.8598$) or women ($P = 0.3819$). However, children had a significantly higher serum ferritin mean in 1989 than in 1988 (98.35 μg/L in 1989 and 59.26 μg/L in 1988; $P = 0.0003$). Comparing the geometric mean of serum ferritin revealed the same conclusions (Table 4). As noted above, all serum ferritin values were normal or elevated. Serum folate means for the 2 years likewise showed no statistically significant difference for men ($P = 0.0753$), women ($P = 0.2112$), or children ($P = 0.9088$). Furthermore, there was no significant difference between 1988 and 1989 serum protein levels for males ($P = 0.9317$), females ($P = 0.0806$), or children ($P = 0.0664$). Vitamin B_{12} values, however, were significantly higher in all subgroups in 1989 than in 1988, even though all

1988 values were normal or grossly elevated (Table 4). While no one test measurement in 1988 or 1989 can definitively confirm the rate of disease in the Kutse population, taken as a group, the entire battery of tests reveals the presence of high morbidity resulting from chronic infectious diseases in both 1988 and 1989.

DISCUSSION

Numerous clinical and experimental studies in both medicine and microbiology have found that the anemia of chronic disease/inflammation is an acute-phase response to a multitude of infectious and inflammatory diseases (Kent, 1992; Kent et al., 1994; Silber et al., 1991; Weinberg, 1978, 1984, and elsewhere). An increasing number of researchers (Basso et al., 1991; Haddock et al., 1991) are suggesting that the activation of the anemia of chronic disease as an acute-phase response is a non-specific reaction of the immune system to pathogen invasion. Hypoferremia, fever, elevated ESR, and normal to elevated iron stores are now known to be produced by various cytokines, particularly interleukin-1 and tumor necrosis factor (Alvarez-Hernandez et al., 1989; Fuchs et al., 1991; Ishii et al., 1991). Similarly, it has been argued (Zang and Chung-Hua, 1990) that hypoferremia following surgical operation may be a defensive mechanism to prevent postoperative infection. The reason hypoferremia is a deterrent to pathogen proliferation is that bacteria and cancer cells require iron but cannot store it themselves in the case of the former, or cannot store enough for propagation in the case of the latter. The body in return attempts to protect itself with a generalized defense by withholding needed iron, accomplished by removing iron from circulation and putting it into storage where it is less accessible (and, hence, the presence of normal to elevated serum ferrin levels associated with hypoferremia in the anemia chronic disease).

When the body is invaded by rapidly proliferating pathogens or neoplastic cells a variety of cytokines are produced (e.g., the interleukins, C-reactive proteins, and others). Some, such as interleukin-1 (IL-1) induce fever, which increases the need of pathogens to extract iron since the elevated temperature causes them to acquire more iron (Weinberg, 1984; Kent et al., 1994). Interleukin-8 (IL-8) is another cytokine produced on exposure to inflammatory stimuli, in addition to interleukin-1 and tumor necrosis factor alpha (TN-α). Interleukin-8 responds to bacterial endotoxins by inducing fever and promoting neutrophil activation and chemotaxis which help fight disease (Zampronio et al., 1994:1670). Interleukin-1 and 8 (in addition to other cytokines as interleukin-6, interferons, etc.) explain the link between fever and disease. They also explain the frequency of elevated temperatures at Kutse, where 50% of those tested had a slight to moderately elevated temperature. Hemoglobin, hematocrit, serum iron, and transferrin saturation levels lower as iron is put into storage, which produces hypoferremia. The cascading consequence results in normal or elevated serum ferritin levels which are an indirect measure of iron storage. Bone marrow aspirated biopsies show adequate or above normal iron stores in the face of subnormal circulating levels of iron, or in other words, the anemia of chronic disease.

High serum ferritin levels have been closely associated with the presence and severity of disease, even in the absence of subnormal serum iron levels (Esen et al., 1991). For example, 32 patients with histologically confirmed renal cell carcinoma had a mean serum ferritin level of 259.1 μg/L (range 5–846 μg/L), in contrast to 22 healthy controls who had a mean of 34.7 μg/L (range 23.9–130.3 μg/L). These figures are significantly different, in spite of the fact that control and patient serum iron concentrations were not statistically significantly different (Esen et al., 1991). In comparison, mean serum ferritin values for 51 Kutse adults in 1989 were not that dissimilar from the ill patients in the above study—219.1 μg/L, with a range of 39–901 μg/L. The Kutse mean and range is also similar to the mean and range of serum ferritin in patients ill with a variety of chronic and acute diseases (Adelekan and Thurnham, 1990; Cash and Sears, 1989; Esumi et al., 1988; Hann et al., 1990; Sakata et al., 1991). Studies show that tumor necrosis factor alpha, which is higher in patients with chronic disease/inflamma-

tion, is negatively correlated with serum iron and transferrin and positively correlated with serum ferritin in the anemia of chronic disease (Vreugdenhil et al., 1992).

ESR is a particularly useful measure for distinguishing iron deficiency anemia from the anemia of chronic disease/inflammation (Beganovic, 1987; Charache et al., 1987; Jansson et al., 1986; Witte et al., 1987). Elevated ESR levels occur in concert with other indicators of the anemia of chronic disease/infections because ESR is an indirect indicator of disease. Its association with disease is partly a result of its strong correlation with interleukin-6, a central mediator of hosts' defense responses to inflammation. Interleukin-6 is a cytokine that stimulates hepatocytes to produce acute phase reactants and B lymphocytes to produce immunoglobulin, resulting in elevated ESR levels (Dasgupta, 1992). There is an inverse relationship in rheumatoid arthritis between hemoglobin and ESR, C-reactive protein, and Clq binding. Tumor necrosis factor alpha was found to be positively correlated with ESR, C-reactive protein, Clq binding, and the presence of chronic disease (Vreugdenhil et al., 1992). The large proportion of individuals with elevated ESR levels at Kutse (42 to 68% of the subpopulations) indicates a high level of morbidity. Normal to elevated serum ferritin levels are also correlated with ESR and C-reactive protein in patients with rheumatoid arthritis (Vreugdenhil et al., 1992). Therefore, elevated ESR, normal to elevated serum ferritin, and elevated temperatures are all interrelated as part of the body's production of cytokines and other acute phase reactants in response to pathogen invasion.

The interpretation that there is high morbidity at Kutse is supported by the 1988 and 1989 serum ferritin data. None of the individuals with low hematocrits had subnormal serum ferritin values. Instead, all serum ferritin levels in all subpopulations were above 15 μg/L both years. We therefore conclude that dietary-induced iron deficiency anemia was not present in the community. The complete absence of any subnormal serum protein values in 1988 or 1989 further supports the view that subnormal hematocrit levels present in this population were not the consequence of an impoverished diet, at least in terms of meat, folates, or vitamin B_{12} (Table 2; also see Appendix). The Kutse diet, adequate in protein and iron, contrasts sharply with other sedentary Kalahari groups, such as those inhabiting Chum!kwe, Namibia, who suffer from documented poor nutrition (Fernandes-Costa et al., 1984).

The level of community morbidity is visible from a number of other hematological tests. For example, elevated protein levels, as an acute phase reactant, varied from 9–10% to 23–29% depending on the subpopulation and year, indicating the presence of infection. Relatively large percentages of each subgroup were associated with either low or elevated lymphocytes, monocytes, and/or eosinophils both years (Table 3). The high levels of vitamin B_{12} found in 1988 and 1989 might result from a genetic variation, although they too are associated with various diseases, such as pulmonary tuberculosis (Morris et al., 1989) and viral hepatitis (Henry, 1984).

Again, these different indices together reflect high rates of disease. In addition, 83% of the camps investigated in 1989 had at least one person with an elevated temperature on the one randomly selected day for monitoring fever in the community (Figure 3). As noted previously, camps vary considerably in size. Windbreak hearths are the social foci of camps. When occupants, including visitors, are together, which represents the majority of the time, people sit very close together and often sleep close to one another around a windbreak hearth. All individuals, whether ill or not, share a common pipe, utensils (if used), and enamel bowls.

The high incidence of morbidity at Kutse in 1988 and 1989, compared to nomadic hunter-gatherers tested in 1969 (Metz et al., 1971), results from the transition to a sedentary, aggregated life-style where outhouses and trash pits are not utilized. As originally proposed by Harpending and Wandsnider (1984), while nomadic and relatively dispersed, Kalahari foragers experienced higher mortality rates because they were subjected to new disease vectors as they moved from camp to camp. Camp sizes were not large enough nor occupations long

enough to build up a reservoir of infectious pathogens necessary for perpetuating chronic disease. Once foragers became sedentary and spent time in larger aggregations, a reservoir was established that encouraged the proliferation and continuation of a heavy pathogen load. In other words, whereas mortality decreased with sedentism/aggregation, morbidity increased, as can be seen in the 1987 Dobe study (Kent and Lee, 1992) and 1988–1989 Kutse studies.

The methods of sanitation that were adequate while people were nomadic are no longer appropriate in the present sedentary aggregated community (e.g., no refuse of any kind is buried). Sedentism and aggregation establish and perpetuate repeated cycles of infectious diseases, resulting in high levels of the anemia of chronic disease, regardless of diet. In areas of endemic bacterial or other infections around the world, anemia has been observed as an effective defense that reduced the frequency and virulence of infections (e.g., Oppenheimer et al., 1986).

CONCLUSION AND PERSPECTIVES

We propose that the 1989 data suggest a high level of chronic disease at Kutse. This interpretation is based on the 1) percentage of individuals with elevated ESR levels and elevated eosinophils, lymphocytes, and serum protein; 2) total absence of subnormal serum ferritin or serum protein levels, including in those persons with subnormal hematocrit values; and 3) clinical observations of a number of ill individuals at a majority of the camps in the community. In addition, 2 months of continuous dietary observations revealed that meat was relatively abundant during this time period and diets were adequate. None of these data demonstrate on their own that infectious diseases are a problem at Kutse, but taken together and coupled with the 1988 data, they provide compelling evidence that there is a high level of disease at this sedentary community.

The shift from a nomadic to a sedentary way of life has been a consistent trend in the Kalahari over the past 20 years. Hematological studies demonstrate a deterioration of health and an increase in the prevalence of disease due to this change in lifestyle. Subsequently, current Kutse morbidity is attributable to the residents' new pattern of mobility and aggregation. When they were nomadic, basic sanitation practices were not essential because people did not occupy one location for long periods nor did they live in large aggregations as they do today. Although the government of Botswana has tried to educate the community on basic hygiene, they have not been very successful in implementing changes. Part of the problem lies in the difficulty of persuading non-Western people with different values and concepts of sanitation that Western hygiene is important for health. A basic obstacle stems from competing systems of knowledge concerning the etiology of disease. For example, it is difficult to convince people about the danger of spreading germs through bodily contact, sneezing or coughing, and other routes, if they do not believe in the existence of germs. The challenge for health care and development personnel is to communicate the importance of sanitation and hygiene in a culturally meaningful way which will be acceptable to non-Western peoples.

Our research demonstrates that it cannot be assumed that low circulating levels of iron are the consequence of an impoverished diet, as is often done in many health studies in developing nations. The first step, which is imperative, is to conduct tests which allow investigators to distinguish dietary iron deficiency anemia from the anemia of chronic disease in order for each to be dealt with appropriately (i.e., measure serum ferritin and ESR levels). Until this distinction is made, medical practitioners, nutritionists, and anthropologists may recommend practices and policies more detrimental than beneficial to the people they want to assist. The indiscriminate dissemination of iron supplements and fortification of foods and salts with heavy doses of iron may hamper the body's nonspecific defense against disease, and raise, rather than lower, morbidity.

We recognize that there are parts of the world where populations suffer from dietary deprivation and require exogenous iron. However, there also are areas where both types of anemia may be operating together. Whereas iron supplementation might be

necessary when bleeding caused by endemic parasitic infections, such as hookworm, is present, in different situations, including those where there is a high prevalence of malaria, oral or intravenous iron will only exacerbate a group's morbidity. Strategies to eradicate both causes must be implemented. Our research reveals that in addition there are areas, such as Kutse and Dobe, where diets are adequate, blood loss due to parasites is minimal or absent, and high frequencies of anemia result from high levels of morbidity. In these cases, iron supplementation would be deleterious to people's health.

After identifying and eliminating the etiologies of chronic disease in these situations, the next step is to devise programs that encourage healthy behavior. The programs' contents should depend upon the target population. Ethnic make-up, cultural beliefs, amount of Westernization (particularly in terms of education and hygiene), indigenous health practices and ideology, individual and communities' particular circumstances, available resources and facilities, and a multiplicity of other factors need to be taken into consideration on a community-specific basis. For instance, although distributing antibiotics alleviates the frequency of some diseases in the short-term, it does not alter behavior, which is the only way to implement changes that will reduce morbidity on a long-term basis. It is unfortunate that we cannot propose specific guidelines which would be relevant for eradicating disease in all communities at risk, but to do so is to miss the point of our article—that morbidity is high for complex and sometimes not obvious reasons. Furthermore, morbidity can only be reduced with methods acceptable to the local people and since anthropologists know concepts of disease and their etiology are culturally constructed, these concepts also vary cross-culturally, as must our methods, in order to be able to incorporate them into community health programs in different societies.

Our research shows that we need to find culturally sensitive and meaningful methods for achieving our goals of improving health, since, as anthropologists, we know that behavior is intricately woven within culture. And, as anthropologists, this is where we can make our unique contribution to the work by physicians and nutritionists who do not possess our knowledge and understanding of culture and its links with behavior, all of which impact health, disease, and individual well-being. Our findings reported here were only possible because of the interdisciplinary nature of the research presented. The challenge, we suggest, is clear, even it the precise methods for specific situations are not.

ACKNOWLEDGMENTS

Primary funding was provided by Trefor Jenkins, M.D., through the South African Institute for Medical Research and the Medical Research Council of South Africa. We acknowledge, with sincere appreciation, our gratitude to the Botswana Office of the President, Ministry of Health, Ministry for Local Government and Lands, and the National Museum and Art Gallery for granting official permission to conduct the health research at Kutse. The Kutse volunteers with whom we worked were both cheerful and patient, and we are most grateful to them for their time, friendliness, and blood. We thank Jane Yates and Trefor Jenkins for conducting the physical examinations at Kutse in 1989, and thank Willy, my field assistant, for interpreting. We also thank Eugene Weinberg and Trefor Jenkins, whose suggestions greatly enhanced this project, Matt Cartmill, editor of AJPA, the anonymous reviewers for AJPA, and the senior author's physician-brother, Steven Kent, for valuable comments on a draft. Denise Elliott and Marian Blue provided important editorial suggestions. Figures were drafted by Old Dominion Graphics Department.

This study is dedicated to Willy, whose short life ended in 1993 because of "sickness." We sincerely hope that interpreting data within an interdisciplinary medical/anthropological perspective will help the other Kutse residents live longer and healthier lives than did Willy.

LITERATURE CITED

Adelekan D and Thurnham D (1990) Plasma ferritin concentrations in anemic children: Relative importance of malaria, riboflavin deficiency, and other infections. Am. J. Clin. Nutr. *51*:453–456.

Alvarez-Hernandex X, Liceaga J, McKay I, and Brock J (1989) Induction of hypoferremia and modulation of

macrophage iron metabolism by tumor necrosis factor. Lab. Invest. *61*:319–322.

Basso D, Fabris C, de Favero G, Meggiato T, Panozzo M, Vianello D, Plebani M, and Naccarato R (1991) Hepatic changes and serum ferritin in pancreatic cancer and other gastrointestingal diseases: The role of cholestasis. Ann. Clin. Biochem. *28*:34–38.

Beganovic S (1987) The erythrocyte sedimentation rate. Ann. Int. Med. *107*:425–426.

Biesele M (1993) Women Like Meat: The Folklore and Foraging Ideology of the Kalahari Ju/'hoan. Bloomington: Indiana University Press.

Burns E, Goldberg N, Lawrence C, Wenz B (1990) Clinical utility of serum tests for iron deficiency in hospitalized patients. Am. J. Clin. Pathol. *93*:240–245.

Cash J and Sears D (1989) The anemia of chronic disease: Spectrum of associated diseases in a series of unselected hospitalized patients. Am. J. Med. *87*: 638–644.

Centers for Disease Control (1989) CDC criteria for anemia in children and childbearing-aged women. MMWR *38*:400–404.

Charache S, Gittlelsohn A, Allen H, et al. (1987) Noninvasive assessment of tissue iron stores. Am. J. Clin. Pathol. *88*:333–337.

Dasgupta B, Corkill M, Kirkham B, Gibson T, and Panayi G (1992) Serial estimation of interleukin 6 as a mesure of systemic disease in rheumatoid arthritis. J. Rheumat. *19*:22–25.

Draper P and Kranichfeld M (1990) Coming in from the bush: Settled life by the !Kung and their accommodation to Bantu neighbors. Human Ecol. *18(4)*:363–384.

Del Beccaro M, Mendelman P, Inglis A, Richardson M, Duncan N, and Shugerman R (1992) Acute-phase reactants and acute bacterial otitis media. AJDC *146*:1037.

Esen A, Ozen H, Ayhan A, Ergen A, Tasar C, and Remzi F (1991) Serum Ferritin: A tumor marker for renal cell carcinoma. J. Urol. *145*:1134–1137.

Esumi N, Ikushima S, Hibi S, Todo S, and Imashuku S (1988) High serum ferritin level as a marker of malignant histiocytosis and virus-associated hemophagocytic syndrome. Cancer *61*:2071–2076.

Fernandes-Costa F, Marshall J, Ritchie C, van Tonder S, Dunn D, Jenkins T, and Metz J (1984) Transition from a hunter-gatherer to a settled lifestyle in the !Kung San: Effect on iron, folate, and vitamin B12 nutrition. Am. J. Clin. Nutr. *40*:1295–1303.

Fischbach F (1992) A Manual of Laboratory and Diagnostic Tests. 4th ed. Philadelphia: Lippincott.

Fuchs D, Hausen A, Reibnegger G, Werner E, Werner-Felmayer G, Dierich M, and Wachter H (1991) Immune activation and the anaemia associated with chronic inflammatory disorders. Eur. J. Haematol. *46*:65–70.

Haddock R, Cousens S, and Guzman C (1991) Infant diet and salmonellosis. Am. J. Public Health *81*: 997–1000.

Hann H-W, Lange, B, Stahlhut M, and McGlynn K (1990) Prognostic importance of serum transferrin and ferritin in childhood Hodgkin's disease. Cancer *66*:313–316.

Hansen J, Dunn D, Lee R, Becker P, and Jenkins T (1993) Hunter-gatherer to pastoral way of life: effects of the transition on health, growth and nutritional status. S Afr J Med Sci *89*:559–564.

Henry J (1984) Clinical Diagnosis and Management by Laboratory Methods. 17th ed. Philadelphia: W.B. Saunders.

Howell N (1979) Demography of the Dobe !Kung. New York: Academic.

Ishii E, Ohga S, Aoki T, Yamada S, Sako M, Tasaka H, Kuwano A, Sasaki M, Tsunematsu Y, and Ueda K (1991) Prognosis of children with virus-associated hemophagocytic syndrome and malignant histiocytosis: Correlation with levels of serum interleukin-1 and tumor necrosis factor. Acta Haematol. *85*:93–99.

Jansson L, Kling S, and Dallman P (1986) Anemia in children with acute infections seen in a primary care pediatric outpatient clinic. Pediatr. Infect. Dis. *5*:424–427.

Johansson J-E, Sigurdsson T, Holmberg L, and Bergström R (1992) Erythrocyte sedimentation rate as a tumor marker in human prostatic cancer: An analysis of prognostic factors in 300 population-based consecutive cases. Cancer *70*:1556–1563.

Kent S (1992) Anemia through the ages: Changing perspectives and their implications. In P Stuart-Macadam and S Kent (eds.): Diet, Demography, and Disease: Changing Views of Anemia. New York: Aldine de Gruyter, pp. 1–30.

Kent S (1996) Hunting variation in a recently sedentary Kalahari village. In S Kent (ed.): Cultural Diversity Among Twentieth Century Foragers: An African Perspective. Cambridge: Cambridge University Press, pp. 125–156.

Kent S and Dunn D (1993) Etiology of hypoferremia in a recently sedentary Kalahari village. Am. J. Trop. Med. Hyg. *48*:554–567.

Kent S and Lee R (1992) A hematological study of !Kung Kalahari foragers: An eighteen year comparison. In P Stuart-Macadam and S Kent (eds.): Diet, Demography and Disease: Changing Perspectives of Anemia. New York: Aldine de Gruyter, pp. 173–199.

Kent S, Weinberg E, and Stuart-Macadam P (1994) The etiology of the anemia of chronic disease and infection. J. Clin. Epidemiol. *47*:23–33.

Lee R (1993) The Dobe Ju/'hoansi. New York: Harcourt Brace.

Metz J, Hart D, and Harpending H (1971) Iron, folate, and vitamin B_{12} nutrition in a hunter-gatherer peopld: A study of the !Kung Bushmen. Am. J. Clin. Nutr. *24*:229–242.

Mohler E (1992) Iron deficiency and anemia of chronic disease: Clues to differentiating these conditions. Postgrad. Med. *92*:123–128.

Morris C, Bird A, and Nell H (1989) The haematological and biochemical changes in severe pulmonary tuberculosis. Q. J. Med. NS *73*:1151–1159.

Nurse G and Jenkins T (1977) Health and Hunter-Gather: Biomedical Studies on the Hunting and Gathering Populations of Southern Africa. Basel: S. Karger.

Oppenheimer SJ, Gibson FD, MacFarlane SB, Moody JB, Harrison C, Spencer A, and Bunari O (1986) Iron supplementation increases prevalence and effects of malaria: Report on clinical studies in Papua New Guinea. Trans. R. Soc. Trop. Med Hyg. *80*:603–612.

Sakata S, Nagai K, Maekawa Y, Kimata Y, Komaki T,

Nakamura S, and Miura K (1991) Serum ferritin concentration in subacute thyroiditis. Metabolism *40*:683–688.

Silber J, Evans A, and Fridman M (1991) Models to predict outcome from childhood neuroblastoma: The role of serum ferritin and tumor histology. Cancer Res. *51*:1426–1433.

Silberbauer G (1981) Hunter and habitat in the central Kalahari desert. Cambridge: Cambridge Unviersity Press.

Vreugdenhil G, Löwenberge B, van Eijk H, and Swaak A (1992) Tumor necrosis factor alpha is associated with disease activity and the degree of anemia in patients with rheumatoid arthritis. Eur. J. Clin. Invest. *22*:488–493.

Wallach J (1983) Interpretation of Pediatric Tests. Boston: Little, Brown and Company.

Weinberg E (1978) Iron and infection. Microbiol. Rev. *42*:45–66.

Weinberg E (1984) Iron withholding: A defense against infection and neoplasia. Physiol. Rev. *64*:65–102.

Witte D, Angstadt D, Davis S, and Schrantz R (1987) Predicting bone marrow iron stores in anemic patients in a community hospital using ferritin and erythrocytes sedimentation rate. Am. J. Clin. Pathol. *90*:85–87.

Zampronio AR, Souza G, Silva G, Cunha F, and Ferreira S (1994) Interleukin-8 induces fever by a prostaglandin-independent mechanism. Am. J. Physiol. *266*:1670–1674.

Zhang X and Chung-Hua W (1990) Hypoferremia following surgery: A defense mechanism after operative trauma. Chinese J Surg. *28*:113–118.

APPENDIX

APPENDIX. Continuous observations of the diet of three families at different camps during the dry season of 1989

Family	Date	Ate meat	No meat
Family 1			
	May 28		X
	May 29		X
	May 30		X
	May 31		X
	June 1		X
	June 2	X	
	June 3	X	
	June 4	X	
	June 5	X	
	June 6	X	
	June 7	X	
	June 8	X	
	June 9	X	
	June 10		X
	June 11		X
	June 12		X
	June 13		X
	June 14		X
	June 15		X
	June 16		X
	June 17	X	
	June 18	X	
	June 19	X	
	June 20	X	
	June 21		X
	June 22	X	
	June 23	X	
	June 24	X	
	June 25	X	
	June 26	X	
	June 27	X	
	June 28	X	
	June 29	X	
	June 30	X	
	July 1		X
	July 2	X	
	July 3		X

(continued)

APPENDIX. Continuous observations of the diet of three families at different camps during the dry season of 1989 (continued)

Family	Date	Ate meat	No meat
Family 2			
	July 4		X
	July 5		X
	July 6		X
	July 7		X
	July 8	X	
	July 9	X	
	July 10	X	
	July 11	X	
	July 12	X	
	July 13	X[1]	
	July 14	X[1]	
	July 15[2]	X	
Family 3			
	July 15[2]	X	
	July 16	X	
	July 17	X	
	July 18	X	
	July 19	X	
	July 20	X	
	July 21	X	
	July 22	X	
	July 23	X	
	July 24	X	
	July 25	X	
	July 26	X	
Total		42	19

[1] The only two days goat meat was eaten. The goat meat was obtained from other camps. On all other days, only wild animals were consumed.
[2] I spent one half of July 15 with Family 2 and one half with Family 3. At each family's camp, meat from wild animals was eaten.